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Prognostic Value of Cardiac Troponin Release in Head Trauma Patients

Thesis

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قالوا

سبحانك لا علم لنا
إلا ما علمتنا إنك أنت
العليم العظيم

صدق الله العظيم

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List of Abbreviations

Abb.	Full term
AIS.....	Abbreviated injury score
AUC	Area under curve
CBC	Complete blood count
CK.....	Creatine kinase
CNS	Central nervous system
CPP.....	Cerebral perfusion pressure
CT	Computed tomography
CTH	CT scan of the head
cTnI.....	Cardiac troponin I
DC.....	Decompressive craniectomy
ECG	Electrocardiography
EDH.....	Epidural hematomas
ELISA.....	Enzyme-linked immune-sorbent assay
FLAIR.....	Fluid-attenuated inversion-recovery
GCS.....	Glasgow Coma Scale
GOS	Glasgow Outcome Scale
ICH	Intracerebral hematoma
ICU	Intensive unit care
LOC	Legth of coma
MC	Monte Carlo test
MI	Acute Myocardial Infarction
MRI.....	Magnetic resonance imaging
mTBI.....	Mild traumatic brain injury
NCIS.....	Neurogenic cardiac injury score
NPV	Negative predictive values
PCS.....	Post-concussive syndrome

List of Abbreviations Cont...

Abb.	Full term
PPV.....	Positive predictive values
PTA.....	Post traumatic amnesia
PTs.....	Post traumatic stress disorder
r.....	Spearman correlation coefficient
ROC	Receiver Operating Characteristic
RTS.....	Revised Trauma Score
SBP	Systolic blood pressure
SDH	Subdural hematomas
SOL.....	Space occupying lesion
sTBI	Severe TBI
TBI.....	Traumatic brain injury
tICH.....	Traumatic intracerebral haemorrhage
Tn.....	Troponin
TnC	Troponin C
TnI	Troponin I

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INTRODUCTION

Traumatic brain injury (TBI) remains one of the main causes of mortality and morbidity among the cases with trauma worldwide (*Dewan et al., 2018*). There is increased incidence of TBI especially in the developing countries with a significant financial annual burden due to more vehicle use. However, in the developed countries, the traffic laws enforcement is associated with a lower rate of TBI incidence related to accidents. However, the incidence of TBI following fall is increasing in these countries, due to the increased number of old people (*Späni et al., 2018*).

The prognosis of patients with TBI is dependent on two aspects, first the damage that happened at the time of initial trauma, and second one occurring during the following days (*Reis et al., 2015*).

Autonomic dysfunction is potentially involved in the secondary damage due to excessive adrenergic activity. This phenomenon leads to heart rate, blood pressure, and thermoregulation abnormalities that may result in myocardial dysfunction (*Tahsili-Fahadan and Geocadin, 2017*).

Although the main etiology and pathophysiology are not well defined, There are several mechanisms regarding relation between the central nervous system and the heart that includes catecholamine release, micro vascular dysfunction, and multi-vessel coronary artery spasm (*Pelliccia et al., 2017*). In these

cases, cardiac dysfunction may vary from asymptomatic conditions to severe myocardial dysfunction (*Pelliccia et al., 2018*).

Publications frequently describe patients with TBI presenting sympathetic discharge signs, including hypertension, tachycardia hyperthermia, and agitation (*Samuel et al., 2016*).

The association between the above conditions and cardiac dysfunction created the concern that a similar relationship could be expected in patients with TBI. Indeed, few studies show that troponin (cTnI) rise after TBI may be associated with bad prognosis. Furthermore, studies show that females with lower cardiac troponin are at a higher risk of cardiac damage compared to males (*Gohar et al., 2017*).

The vast majority of studies focus on evaluating the role of cTnI in coronary artery disease, thoracic trauma, and non-TBI (*Thelin et al., 2017*). Traumatic brain injury (TBI) induce a systemic catecholamine ‘storm’ driven by the central neuroendocrine axis which massively increases sympathetic outflow and activates the adrenal glands (*Gregory and Smith, 2012*).

Inspite of the advancement of knowledge regarding this correlation, sympathetic storm following TBI as the underlying cause of mortality and morbidity in such patients remains not well recognized and undertreated (*Meyfroidt et al., 2017*).

AIM OF THE WORK

The present study aimed at investigating the correlation between serum cTnI levels as a biochemical marker of myocardial damage and morbidity, mortality of the patients with mild to moderate TBI.

Chapter 1

ANATOMICAL CONSIDERATION

Anatomy of neurocranium

The adult human skull is made up of twenty-two bones (*Sadler, 2011*). The cranial cavity, which surrounds and protects the brain and brainstem, is formed by the neurocranium. The occipital, temporal, parietal, sphenoid, ethmoid, and frontal bones make up the neurocranium, which is held together by sutures (*Lang, 2012*).

The brain

In the three dimensional model of the adult brain, there are three clearly identified regions (*Vilela, 2019*):

- Cerebrum: (Telencephalon).
- Brainstem: Mesencephalon (Mid brain), Metencephalon (Pons) and myelencephalon (area that houses the fourth ventricle or Medulla oblongata).
- Cerebellum.

Cerebrum

They are two large oval structures incompletely separated from each other by the falx cerebri (*Yagmurlu et al., 2015*). The surface of each hemisphere is called cerebral cortex